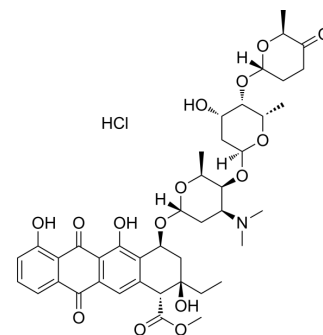


Aclacinomycin A hydrochloride

Cat. No.:	HY-N2306A
CAS No.:	75443-99-1
Molecular Formula:	C ₄₂ H ₅₄ ClNO ₁₅
Molecular Weight:	848.33
Target:	Proteasome; Topoisomerase; Antibiotic; DNA/RNA Synthesis
Pathway:	Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; Anti-infection
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 125 mg/mL (147.35 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.1788 mL	5.8939 mL	11.7879 mL
	5 mM	0.2358 mL	1.1788 mL	2.3576 mL
	10 mM	0.1179 mL	0.5894 mL	1.1788 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.08 mg/mL (2.45 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.08 mg/mL (2.45 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Aclacinomycin A (Aclarubicin) hydrochloride is an orally active and potent anthracycline antitumor antibiotic. Aclacinomycin A hydrochloride is an inhibitor of topoisomerase I and II. Aclacinomycin A hydrochloride inhibits synthesis of nucleic acid, especially RNA. Aclacinomycin A hydrochloride might inhibit the 26S protease complex as well as the ubiquitin-ATP-dependent proteolysis^{[1][2][3]}.

IC₅₀ & Target

Topoisomerase I	Topoisomerase II
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In Vitro

Aclacinomycin A (0-120 μM, 30 min) inhibits the ubiquitin-ATP-dependent proteolytic activity of rabbit reticulocytes in a dose-dependent manner, with an IC₅₀ of 52 μM. But it does not inhibit the ubiquitination^[1].
Aclacinomycin A inhibits ubiquitin-ATP-dependent proteolysis after the conjugation of ubiquitin to proteins^[1].

Aclacinomycin A (0-2.4 μ M, 3 h) inhibits the topo II catalytic activity^[2].

Aclacinomycin A (0-1.8 μ M, 3 h) has negative effect on the proliferative rate of V79 and irs-2 cells^[2].

Aclacinomycin A emits fluorescence and that human-cervical cancer HeLa cells exposed to Aclacinomycin A exhibits bright fluorescence signals in the cytoplasm when fluorescence microscopy was performed using the red filter (excitation 530-550 nm/emission 575 nm)^[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay^[2]

Cell Line:	V79 and irs-2 cells
Concentration:	0, 0.006, 0.12, 1.2, and 2.4 μ M
Incubation Time:	3 h
Result:	Inhibited the topo II catalytic activity in a dose-dependent manner. The loss of topo II catalytic activity in ACLA-treated cells was in all cases significant compared with non-treated cells.

Cell Proliferation Assay^[2]

Cell Line:	V79 and irs-2 cells
Concentration:	0, 0.12, 0.25, 0.37, 0.6, 1.2, 1.8 μ M
Incubation Time:	3 h
Result:	Showed a dose-dependent negative effect on the proliferative rate of V79 and irs-2 cells, but the reduction in surviving colonies was higher in the radiosensitive irs-2 cells for most of the ACLA doses tested.

In Vivo

Aclacinomycin A (0.75-6 mg/kg, IP, daily) dose-dependently exhibits tumor growth in mice-based Leukemia P-388 model^[4].

Aclacinomycin A (0.6-20 mg/kg, Orally, daily) exhibits an antitumor effect on leukemia L-1210^[4].

Aclacinomycin A is very well absorbed in mice, rats, and dogs after its oral administration. The oral LD₅₀ (76.5 mg/kg) is about twice the iv LD₅₀ (35.6 mg/kg) in mice^[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	DBA/2, CDF ₁ (BALB/c \times DBA/2) mice with Leukemia P-388 ^[4] .
Dosage:	0.75 mg/kg, 1.5 mg/kg, 3 mg/kg, 6 mg/kg.
Administration:	Intraperitoneal administration daily for 10 days starting 3 hr after transplantation.
Result:	Inhibited tumor growth.

Animal Model:	CDF ₁ mouse with Leukemia L-1210 ^[4]
Dosage:	0.6 mg/kg, 1.25 mg/kg, 2.5 mg/kg, 5 mg/kg, 10 mg/kg, 20 mg/kg
Administration:	Orally, daily for days 1-9
Result:	Exhibited an antitumor effect on leukemia L-1210.

- Bioengineered. 2022 Feb;13(2):2207-2216.
- Int J Hyperthermia. 2022;39(1):998-1009.
- bioRxiv. 2023 Jan 13.

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- [1]. Isoe T, et al. Inhibition of different steps of the ubiquitin system by CDDP and aclarubicin. *Biochim Biophys Acta*. 1992 Sep 15;1117(2):131-5.
- [2]. Hajji N, et al. Induction of genotoxic and cytotoxic damage by aclarubicin, a dual topoisomerase inhibitor. *Mutat Res*. 2005 May 2;583(1):26-35.
- [3]. Iihoshi H, et al. Aclarubicin, an anthracycline anti-cancer drug, fluorescently contrasts mitochondria and reduces the oxygen consumption rate in living human cells. *Toxicol Lett*. 2017 Aug 5;277:109-114.
- [4]. Hori S, et al. Antitumor activity of new anthracycline antibiotics, aclacinomycin-A and its analogs, and their toxicity. *Gan*. 1977 Oct;68(5):685-90.
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Caution: Product has not been fully validated for medical applications. For research use only.

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